

T H E S I S

For the Degree of Doctor in Medicine,  
Edinburgh University.

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Subject: TOXAEMIA of PREGNANCY.

Etiology and Pathology, with observations on three  
cases of Eclampsia.

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April 30, 1903.

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Cases of Eclampsia.

In the course of general practice I attended three cases of Eclampsia within a period of twelve months and that in a practice of not more than twenty obstetrical cases annually. My treatment of the cases met with very little success. I was led to consider the cause of this serious condition believing that only as we have a definite knowledge of the cause can we hope to lay down a successful line of treatment, which is the grand object of all medical knowledge.

As eclampsia is now generally admitted to be <sup>(u)</sup> but the graver manifestation of a general condition associated with pregnancy, caused by excess of toxic substances in the blood the result of faulty metabolism, or the retention of normal products of metabolism through failure of elimination, it will be instructive to consider the views of physiologists on the subject of metabolism, the products and their excretion. I need make no apology for introducing

introducing into the discussion a few elementary facts, as I find one or two deductions drawn from experiments and clinical observations do not seem to be in keeping with the present day teaching of the physiologist.

It will be impossible to discuss the whole subject of metabolism in the space at my disposal. I shall present a very brief summary of the more important facts which have a bearing on the subject under consideration. I shall demonstrate as far as possible a few of the normal products of metabolism and their source, the organ chiefly concerned in their excretion, also some of the causes that may produce excess of these normal products or lead to the formation of abnormal products.

I shall further compare the physiological effects of these products with the phenomena met with in toxaemia, and hope in a measure to elucidate the etiology of that condition fraught with such risks to the life of both mother and child, and a source of great anxiety to the practitioner in attendance.

Within recent years great additions have been made to our knowledge of the processes that are at work in what we might term the molecular life of the human organism.

organism.

In the individual molecule of protoplasm two processes are going on at the same time, one a building-up process--anabolic, the other a breaking-down process--katabolic. These processes are affected by environment and by nerve action. From whatever reason there is an increase of the anabolic process, there is a relative increase of the katabolic process, with resulting increase of the waste products.

Follow the proteids of the food after ingestion. As a result of digestion they are broken up chiefly into albumoses+peptones, and these are absorbed by the vital action of the epithelial cells of the intestinal mucous membrane, which transform them and pass them into the blood stream as serum albumen and serum globulin. They pass on to the tissues through the liver, no change or storage taking place in that organ. There seems very good reason to believe that the further change of proteids occurs in two ways, either around the living molecule of bioplasm but under the influence of its activity (Voit's theory), or, that the proteids become an integral part of the living molecule and then change. (Liebig, Hoppe Seyler and Pflüger's theory). (24)



theory).

The exact nature of the chemical action that goes on is doubtful; but, from the results we can say definitely that it consists of oxidation and a splitting up with the formation of simpler bodies, part replacing the worn out material of the molecule and part being carried off by the blood stream as waste products. These processes of metabolism are affected by the supply of oxygen and by certain glandular secretions. "It is found that if there be any interference with the normal oxidation of the body generally or of individual tissues by diminished arterial supply or by poisons, probable products of metabolism--lactose and lactic acid--appear in larger amount than usual in the blood and are excreted in the urine." (Schäfer).

The administration of thyroid gland or extract of the gland, or its active principle thyroïdine is followed by a marked increase in activity of the Katabolic process. This action seems to be entirely on fats converting them into carbon dioxide and water. Bleibtren and Wendelstadt also Roos got an increase of nitrogen excreted during the administration of thyroïd, while Richter found in man no marked effect on proteid metabolism.

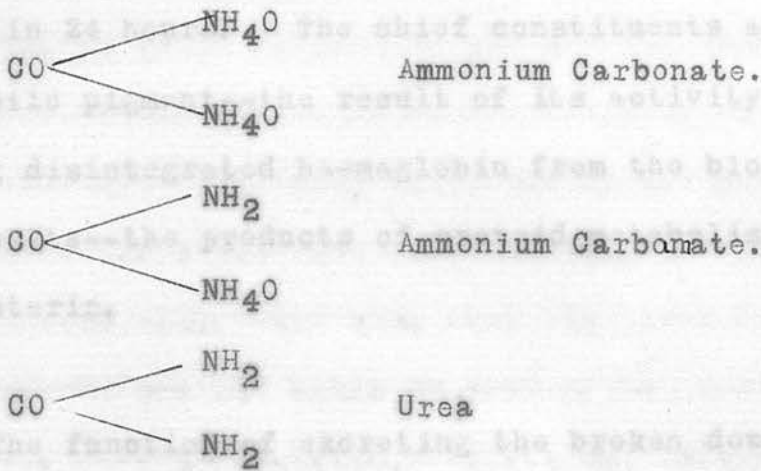
by its secretion--the bile, emulsifying the fats  
metabolism.

At first there is an apparent increase of proteid metabolism indicated by an increase in amount of the urea excreted. Schöndorff<sup>(24)</sup> shows that it is only temporary, and that after nine days the nitrogen balance is maintained, while the fats continue to be greatly reduced in amount. He believes the temporary increase of urea arises from the xanthin bases and the other waste products of metabolism being washed out of the tissues by the water formed, and by the vaso dilatory action of the thyroid. While the thyroid may have no causal relation to eclampsia, this may afford the explanation of the beneficial results got from administration of thyroid extract together with dieting in the cases of eclampsia reported by Dr Oliphant Nicholson.<sup>(17)</sup> The intermediate steps between the proteid of the bioplasm and the great end product of proteid metabolism--urea--are not all known with certainty. I shall return to them in discussing the functions of the liver. The liver has three important functions. It is the chief organ of assimilation in the body.<sup>(10)</sup> It is the great excretory organ on the portal circulation, analogous to the kidney on the general circulation. It also assists to a small extent in digestion by

by its secretion--the bile, emulsifying the fats and thereby facilitating their absorption.

The Function of Assimilation:- The liver converts the glucose--the product of digested carbohydrates--into glycogen, which is stored in the substance of the liver cells and utilised by the system as required. The liver also transforms the products of proteid metabolism into a form in which they are readily excreted. The chief of these intermediate products is undoubtedly ammonium compounds in the form of ammonium lactate; but other bodies such as creatin, uric acid, the allied xanthin bases etc., are also found in muscle. These for the most part are transformed by synthesis into urea in their passage through the liver. Schöndorff (24) ~~and Schröder~~ experimentally demonstrated the presence of ammonium salts. He perfused blood through the limbs of a well-nourished dog and got a marked increase of ammonium salts in the blood. He further demonstrated that the ammonium salts were converted into urea by the action of the liver cells. He perfused blood containing ammonium carbonate through liver, and found it all converted into urea. This change is not found when blood is perfused through other tissues. When we compare the chemical formula, the process is simply that of removing two molecules

molecules of water from the ammonium carbonate.



Indirectly the result of Schöndorff's experiment is confirmed by the experiment in joining the portal and hepatic veins--Ech's fistula--the liver is thus removed from the circulation, and the blood immediately shows a great decrease in the amount of urea and a marked increase in the quantity of ammonium salts. By these experiments it is demonstrated that a diminution in the amount of urea excreted by the kidneys may be the result of failure on the part of the liver to form urea, and not necessarily of failure of elimination on the part of the kidneys.

Fothergill<sup>(6)</sup> says that a diminution in the amount of urea excreted indicates failure of elimination on the part of the kidney. In cases examined by Bernard no increase of urea was found in the blood, although the amount found in urine was diminished.

Excretory function of Liver. Bile is excreted



excreted by the liver in amount varying from  $\frac{1}{2}$  to  $1\frac{1}{2}$  pints in 24 hours. The chief constituents are water, bile pigment--the result of its activity in removing disintegrated haemaglobin from the blood, bile salts--the products of proteid metabolism, and cholesterin.

The function of excreting the broken down haemaglobin is independent of the function of excreting the bile salts; indeed, it is often found that an increase of pigment is accompanied with a decrease of bile salts, as if the rapid disintegration of haemaglobin interfered with proteid metabolism. The liver has the power of arresting alkaloidal substances and certain products of digestion absorbed from the intestinal canal, which if passed into the blood stream would be hurtful to the economy. Schiff<sup>(2)</sup> experimenting with nicotine demonstrated this function of the liver. He found that the dose sufficient to produce death in an animal, when *injected into a peripheral vein does not kill when* injected into the portal venous system of an animal of equal weight. The same dose is fatal if the portal vein has been ligatured, the nicotine passing into the blood stream without having passed through the liver.

liver. ~~It is~~ ~~in 24 hours.~~

Rogers<sup>(2)</sup> using strychnine got similar results.

Bouchard in experimenting on the toxicity of blood, found the blood entering the liver by the portal vein much more toxic than the blood leaving the liver by the hepatic vein, indicating that the liver had either abstracted from the blood ~~and~~ some of the toxic substances, or altered their properties. After the ingestion of food the toxicity of blood is greatly increased while in inanition the toxicity is diminished, and we rightly conclude that it must be due to products absorbed from the intestinal canal. From error of diet, imperfect digestion, or fermentative changes in the bowel, these toxic substances may be in excess or possess irritant properties and force the hepatic barrier, or interfere with the other functions of the liver.

The kidneys are the great excretory organs of the body. Their function is almost entirely that of excretion, and they are incapable of reabsorbing any part of the product they eliminate. They eliminate  $\frac{2}{3}$  of the solid matter--nitrogenous substances and salts etc.,--and a large amount of water from the body. The total amount excreted varies. On an average a man will pass 1,500 c.c.

1,500 c.c., urine in 24 hours.

Though many of the more complex organic bodies in the urine have not been isolated, or their chemical formula ascertained, we can learn much that is of value by considering their physiological action on animals. For our knowledge of this subject we are largely indebted to Bouchard<sup>(2)</sup> and to others who followed his methods. He experimentally demonstrated the toxicity of the various excretions from the body and more particularly that of urine. That urine possessed toxic properties has been known for some time clinically in cases of anuria, producing the chain of symptoms known as uraemia. Bouchard injected normal urine into the vein of a rabbit and obtained the following phenomena in the order given--myosis, increased frequency of respiratory movements, somnolence, diuresis, loss of temperature, coma, and death. The amount required to produce these phenomena varied according to the different specimens of urine. The urine passed during the day or after exertion was less toxic than the urine passed during the night or after rest. The day specimen contained a larger amount of salts, thus proving that the salts are not the cause of the toxicity. Bouchard attributes the decrease in the toxicity to the more complete oxida-

oxidation of the organic products of proteid metabolism. He further carried out a long series of experiments with the different constituents of urine, urea, uric acid, the mineral salts, and other substances five in number, which he separated according to their physical properties, to find if possible the substance or substances that produced these phenomena. Urea induced a marked diuresis. <sup>In larger doses</sup> ~~uric~~ produced death; but the quantity required was ten times greater than that present in the amount of urine necessary to produce death, therefore it cannot be the cause of the toxicity of urine. He likewise excluded uric acid and creatinin. The mineral salts and especially potassium salts produce convulsions; but the quantity required is greater than that in the urine required. Of the five substances which he separated one produced myosis, one salivation, one narcosis, one convulsions, and one reduced the temperature of the body. His results may be summed up in the following; that the toxicity of the urine is due to the combined effects of the urinary constituents, and that the chief cause is the narcotic substance. That the property of one substance may be masked by its association with one of the other bodies, and thus you may have a case showing the convulsive phenomena



phenomena most marked, or a comatose condition most prominent. Bouchard experimentally demonstrates that the kidneys remove poisons from the body, and that suppression of ~~this~~ function would be hurtful. Clinically it is observed that the kidneys are capable of doing far more work than they are called upon to perform in health. They eliminate a larger amount of water and normal constituents and many abnormal constituents without apparent injury to themselves.

I have so far demonstrated that there are substances possessing poisonous properties found in the body and excreted by normal channels, and that these substances may arise from several sources. They may be introduced from without by the food, or arise from the products of digestions or from the putrifactive changes that take place in the intestinal canal. They may result from obstruction to the normal outflow of bile, or from functional inactivity on the part of the liver--the liver failing to arrest the crude products absorbed from the intestinal canal, or failing to transform the products of proteid metabolism. They may arise from excess of proteid metabolism found in the body, or from failure of the function of the kidney owing to dis-

disease, to obstruction, or to imperfect blood supply. In normal health the toxins do not accumulate in the blood; there is a balance maintained between the amount formed and the amount eliminated. That is a condition in which the complex processes of metabolism are perfectly performed, and the elimination of waste products is complete.

In dealing with such a complex mechanism in which each part is dependent upon the other for the proper fulfilment of its function, it is readily seen how difficult it is to isolate the primary cause of any disturbance. It is all the more difficult when we find that similar phenomena are produced by these various toxins which may be present in excess in the blood owing to failure of elimination on the part of the kidney, failure of elaboration on the part of the liver, imperfect oxidation of tissue metabolism, or absorption of abnormal products from the intestinal canal.

With these facts before me I shall now discuss the subject of toxæmia in its relation to pregnancy, now generally spoken of as toxæmia of pregnancy. By this term we understand a condition occurring during pregnancy in which toxic matter is present in the blood in excess. A toxic drug

drug according to the quantity given will produce slight or grave symptoms of poisoning; so with animal toxins, they will produce slight or grave symptoms according to the quantity present in the blood at any given time. Toxines in the blood have been considered the cause of eclampsia--that gravest complication of pregnancy, labour, and puerperium--but there are minor conditions, now considered physiological on account of the comparative regularity and frequency with which they occur, which can be explained as the result of slight toxæmia, and might be safely considered pathological. If these minor phenomena, nausea, vomiting, albuminuria, salivation, headaches, drowsiness, etc., can be looked upon as the earlier symptoms of a toxic condition of the blood and treated as such, by attention to the condition of the excretory organs, then we may expect that the graver phenomenon--eclampsia--will become much less frequent. Garrigues (1902 edition) still speaks of the "morning sickness" as the physiological vomiting of pregnancy. Under no other conditions do we consider nausea and vomiting as physiological. It is more often considered the result of an error of diet, or an important symptom indicating the onset of certain specific

specific fevers. Sir James Y. Simpson taught last

With the onset of pregnancy, and especially with first pregnancies, very important changes take place in the different systems of the body, the blood and the nervous system being chiefly affected. With all growth there is a production of waste material; and as we are now considering not one organism but two in both of which there is rapid formation of new tissue, in the enlarging uterus, and growing placenta and foetus, there will be thrown into the blood stream an additional quantity of waste products. The demands made on the maternal organs will be increased. They have to supply all the nutriment for this rapid growth and eliminate all the waste products. The oxygen necessary for the growth of <sup>the</sup> foetus is taken from the maternal circulation by the foetal blood stream in its passage through the placenta, which in exchange gives the carbonic acid and other waste products, which are ultimately eliminated by the maternal excretories. As most of this material is excreted through the function of the kidneys, attention was first drawn to those cases where kidney failure was the prominent symptom; but there are cases where the kidney is not affected, and we must search for other causes. As



As early as 1841 Sir James Y. Simpson<sup>(22)</sup> taught that patients suffering from puerperal convulsions had almost invariably albuminuria and preceeding oedema, but that all cases of albuminuria did not develop convulsions. "That the morbid condition which leads to albuminuria is usually transitory, from which patients's recovery in a few days, does not depend on any actual structural change of the kidney." "That the oedema albuminuria and convulsions are the effect of one common central cause--a pathological state of the blood to the occurrence of which pregnancy in some way peculiarly predisposes probably from various acts of secretion, nutrition and depuration being vastly increased and altered by the condition of utero gestation."

M. Hippolyte Blot found every case of eclampsia associated with albuminuria and found the albumen entirely disappear within a few days after delivery. None of the patients whom he attended as far as he knew had any indication in after life of a morbid condition of the kidneys, and in the cases he examined after death he found no trace of any renal disease. Consequently although the fashion of his day was to attribute eclampsia to renal disease he considered the belief incorrect and treatment

treatment founded upon that hypothesis must necessarily be wrong. L'Union Medical 1850.

Blot did not attach all blame to the kidney. Sir James Y. Simpson went further and held that the primary cause was an altered state of the blood owing to increased metabolism and the presence of waste products. This was also given as the cause by Dr Matthews Duncan. His opinion was that the condition of the blood was the chief cause of the proneness of women to disease of the kidney and uraemia; and the condition of the glands the further cause of the degenerative changes in liver and kidney. To the same cause he also ascribed the nausea and vomiting of pregnancy.

Ramsbotham (1867)<sup>(21)</sup> stated that the condition of eclampsia originated in a deranged state of the nervous supply to uterus the result of too much albumen in the blood, the immediate consequence of which was irritation propagated from the uterus to the higher nerve centres.

Apart from the interest which is found in the writing of such men as Sir James Y. Simpson, I think this is of special interest when we think of the many theories that have been put forward since then to explain the condition of eclampsia, and that

that we have now returned to their hypothesis, that the cause is a morbid condition of the blood. I do not believe that by that term we mean the same as they did. Our knowledge of the blood condition has increased and the opinions altered, but in the main the hypothesis is still the same.

The condition of the blood in pregnancy was formerly believed to be analogous to that of plethora, and that was the explanation given for many of the characteristic phenomena met with--headache, palpitation, singing in the ear, and albuminuria. The treatment of the condition built on that hypothesis was naturally that of low diet and frequent bleeding of the patient. Sometimes the patient was bled as often as 50 to 90 times during a pregnancy, and Meig believed that if a pregnant woman was affected with headache and that headache coronal it was a sure indication for the use of the lancet, and under such conditions "the lancet and nothing but the lancet was worthy of his confidence." The total amount of the blood in the system is increased and altered. It is more watery. The amount of albumen and the number of red blood corpuscles are diminished, also the haemoglobin but only in proportion to the number of red cells. The fibrin and white corpuscles are

are increased. Dr Oliphant Nicholson draws attention to this condition of the blood as one of the first blood changes which follow thyroidectomy; but this increase of white corpuscles occurs in many inflammatory conditions. Occasionally the condition of the blood may be that of pernicious anaemia when the number of red cells is diminished and the cells present are irregular in shape and size ~~and~~ - poikilocytosis.

Bouchard proved the toxicity of the blood serum of a healthy individual. He demonstrated that the blood serum of a dog when injected into rabbit produced death, that this was not due to mechanical means, as salt solution produced no effect, unless in much larger doses.

Doleris<sup>(8)</sup> in 1886 found that the toxicity of blood in albuminuric cases was increased.

Tarnier and Chambrelent<sup>(9)</sup> confirmed this observation and further found that the blood of patients suffering from eclampsia had double the toxicity of the blood of a healthy pregnant woman. These observers (Tarnier and Chambrelent) maintained that the toxicity of the blood was in inverse proportion to the toxicity of the urine; if blood more toxic, then urine less toxic.



toxic.

Ludwig and Savor<sup>(8)</sup> in their experiments on the toxicity of blood serum found that the blood of a pregnant woman was more toxic than the blood of a nonpregnant woman, and that the blood of a eclamptic was more toxic than that of a healthy pregnant woman. These observers all agree that in pregnancy the toxicity of blood serum is increased, and that is what we might expect from the increase of waste products thrown into the blood stream.

Volhard<sup>(28)</sup> while believing in the toxicity of the serum and urine throws doubt upon the increase of the toxicity in pregnancy. There are many difficulties in the way of estimating the relative toxicity of the serum of pregnant and nonpregnant women, and of pregnant and eclamptic women. The toxicity of the blood varies in different individuals, and varies with the quantity and quality of the food given, and with the amount excreted by the different organs of elimination. We should require to test the toxicity of the blood and urine in a nonpregnant woman, and then again the same in the pregnant condition; and very rarely would any of these develop eclampsia so as to enable us to compare eclamptic serum with that of the pregnant and nonpregnant

nonpregnant conditions in the same individual.

Along with this altered condition of the blood there is increased arterial tension and hypertrophy of the cardiac muscle. Allbutt<sup>(v)</sup> compares the phenomena with that met with in cases of chronic lead-poisoning, and believes that the increased arterial tension is the result of toxins circulating in the blood. This is well exemplified in chronic cases of albuminuria where the high tension pulse, thickened walls, and cardiac hypertrophy are well known features. A fatty degeneration of the heart, especially of the papillae may be seen.

In a large percentage of pregnancies the urine shows the presence of albumen. The frequency varies according to different writers. In the earlier months Herman gives it as low as one per cent. Blot gives it as high as 20 per cent but the average taken from the records of Ingersley, Meyer, Saft, and Schroeder is about 4.25%. As pregnancy advances the number of cases showing albuminuria increases, until, in the last month, the percentage is given as 82 by Fischer, 71 by Meyer and 54 by Saft.

The quantity of urine passed may vary considerably, but it is usually increased with a low specific gravity. Chlorides show increase while phosphates,

phosphates, sulphates, urea and uric acid show a decrease. Tube casts are rarely present.

Although the presence of albumen always calls for careful investigation it is not of so much importance as the estimation of the quantity of urea excreted. In eclampsia or with the onset of premonitory symptoms the amount of albumen in the urine may be very small, or it may be present in a large amount; but the amount of urea found is constantly much less than normal. Davis<sup>(5)</sup> reports several cases where the only symptoms were epigastric pain and frontal headache. The urine contained no albumen or only a trace; but there was a marked diminution in the urea output.

<sup>(5)</sup> In 564 examinations of the urine of 84 cases the average percentage of urea present before labour was 1.4 while after labour it was 1.9. In two cases immediately before an attack of eclampsia the urea was .5%, on the day following it had increased to 2.4.

Davis considers that the amount of urea is valuable as an index of the excretory power of the kidney. It is probably of more value in first estimating the condition of the urea forming function of the liver. Owing to the diminution of urea, which by

by Bouchard was found to possess very marked diuretic properties, the excretory power of kidney will be impaired.

I have previously mentioned that Bouchard demonstrated that normal urine possessed poisonous properties. This has also been proved by Charpentier.

Lanlanie and Chambrelent<sup>(8)</sup> found the toxicity of urine of healthy pregnant women less than the urine of healthy non pregnant women. Ludwig and Savor, experimenting with the urine of 8 healthy women in labour and the urine of 16 eclamptic cases, found that the urine of the eclamptic cases was less toxic than that of the healthy pregnant women, and that after the attack of eclampsia the toxicity of the urine increased. Tarnier and Chambrelent found the toxicity of urine greatly diminished in cases of eclampsia. Blanc<sup>(18)</sup> experimenting with the urine passed in the 8th and 9th month of pregnancy also found it less toxic than that of health, and that during the puerperium the toxicity was quite equal to that of health. Volhard showed that the toxic properties of urine of pregnant women varied greatly in amount.

Stewart<sup>(23)</sup> in 1897 experimenting with 12 specimens of urine of pregnant women and 4 specimens of urine of



of non-pregnant women found the urine of pregnant and non-pregnant--virgin and multipara--had about equal toxicity. He collected the specimens in a clean jar to which was added a little Boric acid, and excluded as far as possible the effects of bacterial infection. In more recent experiments 1900, he drew off and collected the urine under aseptic conditions and found the toxicity of healthy urine less than that given by Bouchard, and that it increased with the time which elapsed between collecting and injecting into animals.

The results obtained by Volhard and Stewart throw doubts on the accuracy of the results obtained by the other observers, the results of the latter being in their opinion partially due to foreign material in the specimens used.

If the healthy products of metabolism have toxic properties we should find in the condition of pregnancy an increase of the toxicity of blood serum and an increase in the toxicity of urine. In a healthy physiological condition a perfect balance is maintained between the amount of waste products formed in the body and the amount excreted. If the urine in a healthy pregnant condition is less toxic than the serum, it indicates either a failure of the

the excretory organs or the presence of abnormal products of metabolism in the blood which are in an unsuitable form for elimination by the kidneys. We have seen that the presence of abnormal products in the blood could be obtained by removing the liver from the circulation by means of Ech's fistula, and the result was death of the animal.

The kidney is found affected in from 90 to 95 per cent of the cases of eclampsia,<sup>(11)</sup> and this morbid condition has been carefully studied. The most frequent condition met with is that known as pregnancy kidney, which has been defined by Saft<sup>(8)</sup> as "a disease of the kidney which takes rise in pregnancy alone, never leads to important disturbances of the general health and in child bed quickly subsides". A few cases show evidence of acute or chronic nephritis the same as may be met with in non pregnant conditions.

Morbid anatomy. The kidney may vary in size, and be pale, red, or granular. They have been classified into three groups according to the conditions found on post mortem examination.

1. The normal kidney with no sign of disease and Schauta found out of 70 cases 7 normal.

2. Kidney showing the morbid changes got apart

apart from pregnancy, acute or chronic nephritis, hydronephrosis, dilatation of ureter and pelvis.

3. Pregnancy kidney--the condition showing degenerative changes of the epithelium lining the tubules in the cortical portion. Leyden believed it to be a fatty infiltration. The changes are not those of inflammation, nor are they the result of backward pressure. There is no infiltration of leucocytes. Cohnheim<sup>(27)</sup> draws attention to the anaemic condition, and believes it to be the result of spasm of renal arteries. The authorities quoted by Herman all maintain that it is a true degenerative process attributable to toxic matter in the blood. Virchow found fat emboli in the kidney possibly secondary to the fatty degeneration found in the liver cells. The same degenerative changes are seen in the foetal kidney.

A few indications of the presence of toxins in the blood may be seen in the alimentary system. Nausea and vomiting, though of very frequent occurrence in the early months of pregnancy, is not a necessary accompaniment. Giles states that 33% of women go through pregnancy without having nausea and vomiting. It usually begins about the end of the first month, but it has been seen as early as first week.

week. It is usually very slight occurring in the morning on getting up and gives rise to no great discomfort. It generally passes off by the end of the third month.

Occasionally it persists, or it may disappear to return about the 6th month in a severe form. All food is rejected, tongue becomes dry and furred, gums spongy and bleeding, breath offensive, urine scanty and high coloured, pulse rapid, expression pinched and anxious, skin cold and clammy--a very typical picture of the clinical symptoms seen in many cases of poisoning.

The condition may be accompanied by abdominal pains, diarrhoea, headache, unpleasant odour from skin, delirium, and coma. This is a very extreme condition and not often met with. There is really no difference between the slight nausea and vomiting seen in the early period of pregnancy and the severe form with uncontrollable vomiting in the later months beyond that of degree.

The slight form is usually attributed to reflex irritability set up by the enlarging uterus and is often associated with uterine displacements and catarrh of cervix. Klein considers it to be entirely neurotic and hysterical. No doubt it is



is most frequently seen in patients of a very nervous temperament; and, although this may be a sufficient explanation of the minor forms of nausea and vomiting, it cannot be sufficient for the severer, which in most cases is but a continuation and exacerbation of the early form. In hysterical vomiting the only symptoms seen is that of inanition and only when the vomiting has been going on for a lengthened period.

That other causes may be at work affecting the nervous system is well seen in the necropsies of two cases of uncontrollable vomiting reported by Lindeman and Stembo.<sup>(48)</sup> Stembo found neuritis in several nerves and deterioration of heart similar to that seen in toxic conditions. Lindeman found marked neuritis specially marked in the phrenic nerves and a fatty degeneration of liver and kidney of both mother and foetus. All the conditions were very characteristic of toxine poisoning.

Associated with nausea, excessive salivation may be present. This may be the result of the retention in the blood of the substance isolated by Bouchard from the urine--which has sialogenous properties. We have good reason for this supposition from the fact that the only treatment found benefi-

beneficial is such as has a stimulating effect on the excretory functions of the kidney. Astringents have no effect.

The same may be seen in the treatment of nausea and vomiting. The usual remedies which are found useful in ordinary vomiting, are of little use in checking the vomiting associated with pregnancy.

Constipation is the most frequent condition met with in pregnancy, and from the retained fecal matter absorption takes place of the products of decomposition. The liver will in the ordinary course arrest these toxic substances and return them to the bowel; but in time, if not carried off by the bowel, they may accumulate and force the hepatic barrier.

Sir Lauder Brunton<sup>(3)</sup> considers that it is failure of the function of the liver to abstract the products of intestinal absorption that produces the chain of symptoms associated with inactivity of liver, bitter taste in mouth, headache, depression, and cloudiness of intellect. The normal products of digestion are non irritating but abnormal products arrested and discharged by the bile may produce an irritative effect on the epithelial lining of the bile ducts, cause retention of bile, and thus indirectly interfere

interfere with the glycogenic and urea-forming functions of the liver.

Dr Dewar<sup>(4)</sup> reports a case of eclampsia at the 6th month of pregnancy where the chief condition was constipation. There was no albumen found in the urine.

Dr Savory<sup>(26)</sup> reports a case where a second non-fatal attack of eclampsia was accompanied by haemoglobinuria and constipation. Unconsciousness lasted nine days but was suddenly regained after mechanical means had been adopted to empty the bowel of fecal matter. Dr Kynoch<sup>(13)</sup> reports a case of acute mania coming on during the puerperium. Urine was found healthy, and after free purgation the acute symptoms disappeared. These facts are sufficient to show the important bearing constipation and the contents of the bowel have in the production of toxæmia, either directly by the absorbed material passing into the blood stream, or more probably indirectly, by first irritating and interfering with the normal functions of the liver, whereby imperfect products accumulate in the blood and affect the higher nerve centres, producing irritability of temper and lassitude, melancholia, and mania.

The liver in some cases is the primary organ affected. This is seen in its extreme form in the

the condition known as acute yellow atrophy. Dr Kynoch<sup>(13)</sup> reports the case of a woman suffering from headache and vomiting at the 7th month of pregnancy. She was vomiting bilious matter, tongue was thickly coated and breath offensive. There were no signs of labour and foetal heart sounds were distinct. Urine was abundant--Sp Gr 1020: no albumen. Bowels acting freely, no jaundice but a slight increase of hepatic dulness. She was delivered of a premature dead child the following day. Patient was very drowsy, became restless and died comatose six days after delivery. Post-mortem the liver was large and showed fatty degeneration at the periphery of the lobules. The kidney showed no sign of disease. There had been no failure of kidney function. The amount of urea present in the urine is not given.

Van Santvoord<sup>(27)</sup> reports notes of a case he had observed in two pregnancies. A trace of albumen was present in the urine in the first. Pregnancy was normal. In the second pregnancy she had sudden loss of consciousness followed by headaches, spots before the eyes, and pain in loins followed by loss of vision. The amount of urea was as low as 8.6 grammes or about  $\frac{1}{3}$  of the normal for 24 hours.

The patient ate well, meat with one meal a day, so



so that the diminution was not due to diet. The quantity of urine was normal and contained no albumen. The function of the kidneys seemed perfect the only feature was the diminution of urea.

According to Murchison<sup>(15)</sup> in acute yellow atrophy, a condition met with in pregnancy, the urea is diminished or may entirely disappear from the urine. Leucin and tyrosin may be present but are not constant. In these cases the products of metabolism are allowed to accumulate and circulate in the blood and produce the toxic phenomena.

Practically all obstetrician lay great stress on the quantity of urea excreted. It is found that in all cases where the urea is far below normal the premonitory systems of eclampsia--headache and gastric pain--are usually present, and unless remedied an attack of eclampsia may supervene. No excess of urea has been found in the blood in cases of eclampsia. Therefore the failure must be of the urea forming function of the liver.

The other functions of the liver are frequently seen at fault; there may be slight jaundice, or the glycogenic function may fail, when there is glycosuria. Here it may be interesting to recall the beneficial effect of morphia on cases of glycosuria.

glycosuria. It or its allies are the only drugs which control the amount of sugar excreted by the urine; and may this not be the explanation of the beneficial effects of morphia in cases of eclampsia?

It is only within recent years that the morbid anatomy of the liver in connection with eclampsia has received special attention. The colour of the liver is paler than usual as the effect of epithelial changes. There are numerous hæmorrhages of varying size on the surface and in the liver substance. Microscopically these present a variety of conditions--a large central area of necrosis showing various stages of disintegration of the dead liver cells and blood corpuscles, vessels, surrounded by a ring of engorged capillaries, and small celled infiltration. From these areas Virchow believes that fat emboli pass to other organs.

Small hæmorrhagic areas undergoing degenerative changes, similar to those seen in the liver and kidney may be found in spleen, liver and pancreas.

It is well known that certain toxines have a special action on the nervous system; and many of the more important conditions observed--headache, drowsiness, cloudiness of intellect, irritability of temper, depression, delirium, coma and neuritis--have already

already been referred to.

The brain may show minute haemorrhages in various parts either at the base or in the ventricle, sometimes marked oedema with flattening of the convolutions.

The foetal organs show the same pathological lesions as found in the maternal organs, and the same changes in blood and urine.

The morbid conditions seen are many and varied and it is extremely difficult to say whether they should be looked upon as cause or effect, but the variety and the extent of the lesions can only be explained by a general cause--the presence of toxic material in the blood.

Many of these minor symptoms are seen in a large percentage of the cases of pregnancy. Rich and poor suffer alike. Primiparae suffer more than multiparae and cases of plural birth more than single.

Many theories have been put forward from time to time and have been received with a certain measure of approval but they all fail to explain fully the variety of conditions met with.

1st. The Neurotic Theory. The advocates of this theory maintain that excessive stimuli are set up in the uterus which can act reflexly on the renal nerves

nerves causing albuminuria, on the ~~sympathetic~~ <sup>gas</sup>tric nerves causing nausea and vomiting, and on the higher nerve centres producing headache, irritability, delirium, convulsion and coma.

Beyond the fact that it is seen most frequently in women of a very nervous temperament there is little to support this theory.

2nd. Bacterial Theory. The chief supporter of this theory is Stroganoff<sup>(25)</sup> who believes that eclampsia is due to an unknown organism, and that the incubation period is from 5 to 20 hours and that the infection lasts for three weeks. Bar and Renon and Lensden<sup>(1)</sup> found no evidence of bacteria in the blood either before or immediately after death. Eclampsia is never epidemic and is chiefly seen in primiparae.

3rd. The Toxaemic Theory. That the condition is due to a toxine--it may be one or more--the nature of which is still unknown. This covers ~~most~~ <sup>most</sup> of the cases met with and is the most generally accepted theory. There is still a great diversity of opinion as to the nature of the toxine and its source.

As the kidneys were the organs chiefly affected the cause was attributed to retention of one or more of the urinary constituents.

Fr~~ie~~richs<sup>(11)</sup> believed that it was due to retention



retention of urea or its product carbonate of ammonia--in the blood.

Bouchard's<sup>(2)</sup> experiments proved that urea was not the cause of the toxicity of normal urine. Christison and MacLagan<sup>(2')</sup> and Bernard found no increase in the amount of urea in the blood, and Hamilton<sup>(5)</sup> proved that no such change as the transformation of urea into Carbonate of Ammonia took place in the blood.

It was then attributed to retention of all the normal constituents of the urine owing to failure of function on the part of the kidney--a case of uraemia. This is supported by the following facts:- that normal urine has toxic properties (Bouchard); that the urine in pregnancy is less toxic than the urine in non pregnant condition; that in eclampsia the amount and the toxicity of urine is much less than the urine in healthy pregnancy; and that when recovery takes place there is a great increase in the amount of urine passed and its toxicity is equal to that of health.

Physiology teaches that the constituents of urine are abstracted from the blood and not formed by the kidney, that any deficiency of its toxicity means that the toxins are retained in the blood. Therefore in cases of eclampsia there must be a great

great increase of the toxins in the blood.

Apart from the doubts thrown by Volhard and Stewart on the assertions of Tarnier and Chambrelent and others that there is diminished toxicity of urine in pregnancy, and the fact that in many cases the kidneys functionate normally and at post mortem show no sign of disease, there are still the lesions in liver and brain which cannot be explained by this theory.

The failure of function on the part of the kidney has been attributed by some observers to the effects of pressure. The enlarging uterus by direct pressure obstructs the return of blood through the renal veins or causes retention of urine by pressure on the ureters or by the increase of the general intra-abdominal pressure. This theory is supported by the frequency in multiple pregnancies and in primiparae, and during the later months when the pressure is greatest. Bartel showed that direct pressure on the renal veins was anatomically impossible; the condition found in kidney is not that resulting from backward pressure, but is a degenerative change. Obstruction of the uretus is seen in very few cases. Such a condition as eclampsia is never seen in cases of very large abdominal tumours.

tumours.

Stumpf<sup>(1)</sup> held that the poison is a product of abnormal decomposition in either mother or child, a non-nitrogenous substance with the properties of acetone which in its excretion produces an irritation of the kidney resulting in nephritis; that this body has a destructive effect on the colouring matter of the blood and on the liver cells, leading to degenerative changes and interference with its normal function, causing glycosuria and the appearance of leucin and tyrosin in the urine, and inducing coma and convulsions from irritation of the brain. Fehling lends support to this view, and holds that albuminuria is but the early sign of toxaemia. Schmorl believes that this abnormal product arises from the placenta. The fact that the condition is more frequent in twin births, and that the death of the foetus or emptying of uterus gives a more favourable prognosis for the mother, lends support to this theory.

The last theory is associated with the name of Bouchard and may be called the Auto-intoxication Theory. Bouchard believes that the toxins result from failure of the functions of the liver as well as failure of the function of the kidneys, and that the effects are due to accumulation in the blood not only

only of urinary substances but biliary substances and ptomaines from the bowel which are no longer destroyed in the liver.

(II)  
Bouffe de Saint Blaise believes that it is chiefly the effect of toxines formed in the intestine and carried to the liver, and that the haemorrhagic areas in the liver are pathognomonic.

(II)  
Auvard and Riviere include the failure of elimination on the part of the lungs and skin, i.e., the toxines are present in the blood owing to failure on the part of all the excretory organs.

In an earlier part of this dissertation we found Bouchard proved four possible sources of infection, the food and the products of the fermentative changes in the bowel, the biliary excretion, the products of metabolism, and the normal constituents of urine.

In pregnancy some of the most frequent conditions met with--dyspepsia and constipation--favour the formation and reabsorption of the abnormal products from the bowel; and from the notes of cases reported there can be little doubt that this is a source of infection. For a time these products may be arrested by the activity of the liver function and returned to the bowel; but soon evidence is given that these



these products are passing into the blood stream by the headache and drowsiness which follow. Bouchard found that bile had greater toxic powers than urine, and retention of bile produces the well-known phenomena associated with jaundice.

But we found that the liver had two other important functions, the glycogenic function, failure of which is readily recognised by the presence of sugar in the urine; and the urea forming function, transforming the waste products of proteid metabolism into urea. Failure of this function allows these waste products to accumulate in the blood and set up the various symptoms. This was found as the result of removing the liver from the circulation by joining the portal and hepatic vein.

The chemical action which takes place in the process of proteid metabolism consists of oxidation. If from any cause the supply of oxygen is deficient abnormal products are found in the blood, but with complete oxidation there is an increase in the amount of carbon dioxide excreted by the lungs and a diminution in the toxicity of urine. I consider this a very important point in connection with toxæmia, and its <sup>indirect</sup> ~~real~~ cause might be ascribed to the evils of civilisation--deficient exercise and excess

excess of nutriment. During pregnancy patients as a rule lead a very sedentary life, their appetite is good and they possibly eat more than at other times, and take less exercise. I have observed this among all classes. This must induce a sluggish action on the part of all the organs of the body and consequently the general health is below par. It will be of interest to summarise the results of experiments on the effects of gentle exercise on the body, obtained by Pettenkofer, Voit, Parkes, North, and others as reported in Notter and Firth's Hygiene.<sup>(19)</sup>

The chief effect of exercise is to increase oxidation, the amount of oxygen inspired and the amount of carbon dioxide expired is increased by about 40%. The heart's action is increased in force and frequency, the flow of blood is augmented, more nutriment is carried to the tissues, and the waste products are more quickly removed. Digestion is more perfect, absorption more rapid, and abdominal and hepatic circulation is more vigorous. The skin acts more freely; the water salts and acid pass off in greater abundance. The amount of urine is lessened; water and salts diminished owing to the evaporation of sweat; the urea is slightly increased, and the toxicity of urine is diminished (Bouchard).

(Bouchard). It acts as a safeguard against the

If exercise is deficient nutrition suffers and a degenerative process sets in. The heart's action is weakened and fatty degeneration sets in. The appetite and digestive powers are lessened and the skin is less active. There is a heightened sensitiveness of the nervous system, a morbid excitability and a greater susceptibility to the action of external agencies. Briefly it indicates that all the organs are below par, and in pregnancy that occurs under circumstances where greater demands are made upon them.

As pregnancy advances to full term the maternal organs must supply an increasing amount of nutriment for the growth of the enlarging foetus, and must remove from the blood stream an ever increasing amount of waste products. All the organs have extra work thrown upon them and consequently there are more frequent signs of toxæmia in the later months. These symptoms will be more marked in twin pregnancies where there are two foetuses to be provided for, and in very young primipara where the maternal organs have not reached a state of maturity and are less able to bear extra strain.

The condition of the liver is of primary import-

importance. It acts as a safeguard against the introduction into the system of toxins from without; it also acts on the toxins formed in the process of metabolism, removing them from the blood stream or altering their composition, and forming bodies which can readily be removed by the other organs of elimination.

If from any cause the normal activity of the liver is interfered with--and this most frequently occurs as the result of error of diet and deficient exercise--abnormal products pass into the blood stream. In the course of the circulation they will be carried to the kidneys where they will be removed from the blood. These bodies, in their passage through the renal epithelium, will cause injury to the cells and allow of the transudation of albumen into the urine.

The liver failing to transform the products of proteid metabolism into urea, there will be diminished urea, and the function of the kidneys will therefore suffer for lack of the diuretic action of the urea; and we shall have increase of abnormal products as the result of liver failure, and retention as the result of renal failure.

That the kidney is not the essential organ at



at fault is proved by many cases with renal disease passing through pregnancy without any marked symptoms of toxæmia.

In a few acute cases of toxæmia the liver alone is affected; and owing to its larger blood supply the amount of toxins present at any given moment must be greater than the amount present in the kidneys with their lesser blood supply, and cause rapid destruction of liver cells, and prove fatal before the toxins have had time to destroy the renal epithelium. With our knowledge of the possible sources of toxic substances we can see that by attention to diet and the state of the alimentary tract, and by facilitating the complete oxidation of the products of metabolism, we can lessen the amount of work required of the liver, and have a very efficient means of guarding against the formation, absorption, and retention of abnormal products.

The apparent cause of toxæmia must vary in different cases; but the essential cause seems to be due to an abnormal product of proteid metabolism resulting from insufficient oxidation, or from failure of the urea forming function of the liver--the one feature constantly present is the marked diminution in the amount of urea excreted with no increase of the

the amount of urea in the blood. The failure of the liver function may result from excess of metabolic products thrown into blood stream by the enlarging uterus, placenta and foetus, or from absorption of abnormal products from the intestinal canal, which injure the liver cells and interfere with their normal functions.

Labour pains began on the evening of Nov. 17th, continued during 18th, pains very severe and frequent, a sealing foetal membrane was born on the morning of the 19th.

She was attended by midwife; placenta came away readily and there was very little hemorrhage. Patient felt very drowsy but all stomach well and she was quite comfortable. About five hours later without any warning she was seized with convulsions, which came on every twenty minutes, more frequently growing out of and into another. I saw her two hours after first fit. She was a strong muscular woman, there was no swelling of feet or legs. Fits began with twitching of eyelids and oscillation of eyeballs, face was drawn to left side, limbs and chest spasms of all the

1st Case.

Mrs.                      aet. 23 years.      Primipara.      She had been married 5 months, husband was a milk salesman.      She had comfortable home and enjoyed good health in general with occasional headaches.      The night before labour commenced she attended the local fair and returned in a very excited condition.

Labour pains began on the evening of Nov. 17th, continued during 18th, pains very severe and frequent; a healthy full time child was born on the morning of the 19th.

She was attended by midwife; placenta came away readily and there was very little haemorrhage. Patient felt very drowsy but all seemed well and she was left comfortable.      About five hours later without any warning she was seized with convulsions, which came on every twenty minutes then more frequently passing out of one into another. I saw her two hours after first fit.      She was a strong muscular woman, there was no oedema of feet or legs.      Fits began with twitching of eyelids and oscillation of eyeballs, face was drawn to left side, tonic and clonic spasms of all the

the muscles, frothing at the mouth, the tongue was lacerated, pupils were dilated, pulse 120 regular in time, tension very high, breathing irregular, slow and laboured. Patient never regained consciousness after first attack and died in twelve hours.

I first gave Hydrarg. Subchlor. gr V. followed by Ammonium Bromide and Chloral Hydrate of each gr. 15, and used hot pack. Skin remained dry and harsh. Two hours later pulse was 160 very irregular and small with high tension, breathing very slow and laboured, face livid and no abatement of "fits." I then administered chloroform and continued to do so for over an hour. This lessened the severity of the fits but not their frequency. Temperature 103° F. I then opened superficial vein in arm and drew off a pint of blood which was of a very dark colour. After removal of blood the face was less livid, pulse 140 per minute less irregular, tension diminished and breathing more regular. The improvement was but temporary and patient died as above stated. There was no action of bowels, and no urine passed. The skin remained hot and dry. The child is



is healthy and strong, not subject to convulsions.

No post mortem examination was allowed.

She was in 6th month of pregnancy. There were no labour pains, no rigors, no vomiting brought

### 2nd Case.

Mrs. She was 21 years of age. Primipara.

Husband a labourer 22 years. She had been a strong healthy girl; married only 3 months. Her home was poor, far from comfortable. On Jan. 11th, soon after tea she complained of aching pains in back, and at 9 p.m., feeling very tired she went to bed. While undressing she said "she was getting blind." On getting into bed she had a slight fit and had four more before midnight. I saw her soon after midnight; she was lying in a drowsy condition, breathing rapidly, eyelids closed, pupils contracted, pulse 140 per minute irregular, tension very high, could detect no thickening of vessel walls, heart sounds pure. Patient was well nourished. There was no oedema and no ascites, no pallor and no cyanosis. Convulsions began by raising of eyelids and rolling of eyeballs to right side, head drawn to right side, spasms passed down right arm, then right leg, left

left arm and left leg passing off in the same order.

She was in 8th month of pregnancy. There were no labour pains, os rigid; examination brought on convulsions.

She was given Calomel gr V. and Chloral Hydrate and Ammonium Bromide gr. 15 same as previous case. This controlled the severity of the fits for a few hours. Hot pack was also used. After a consultation early in the morning it was decided not to empty uterus. There were still no labour pains.

At 10 a.m., temperature was 104 F. Pulse 156 very irregular and unequal, tension high, respiration 60 per minute. Catheter passed and about 1 oz., urine drawn off. Enema of soap and water was given and bowels acted freely, and injected hypodermically 1/100 gr., Pilocarpine, this seemed to produce no effect. Hot Pack continued. Fits increased in frequency and in severity, patient remaining comatose.<sup>ar</sup> 3 p.m., a small quantity of urine<sup>had been</sup> passed in bed. Pulse 190 per minute very unequal, breathing slow and laboured. Slight moisture of skin but still very hot. Patient

Patient died soon after.

Urine was pale in colour with heavy white deposit. Albumen very abundant. Urine kept for 6 months without undergoing any apparent decomposition.

Her sister had noticed the patient was very puffy under the eyes for quite a week previous. She had also noticed that the urine was very scanty and of a whitish colour. Patient had been very despondent for some weeks and always remained indoors. Husband had not been good to patient. He had not spoken to her for some weeks and she worried over this very much.

### 3rd Case.

Mrs H                      aet, 28 years.      Multipara.

Husband an engineer's fitter. She had been married 6 years and had borne two children. She had a comfortable home in an open district and plenty of good food.

In first confinement the delivery was by forceps and the puerperium normal. Child died at 18 months from Tubercular peritonitis. In the

the second confinement, delivery and puerperium were normal, child healthy and not subject to convulsions. In both pregnancies she had suffered from oedema of feet and legs and very bad varicose veins. During the third pregnancy she suffered from severe nausea and vomiting during the first month. This soon passed off but returned about the sixth month and continued at intervals during the rest of the period. At the same time she suffered from very severe occipital headaches, puffiness of the eyes, sometimes being unable to raise the eyelids in the morning, and marked oedema of feet and legs. She said urine was very thick but not diminished in quantity. Bowels were constipated.

Labour pains began at 5 a.m., strong and regular, but about noon they became very short and almost ceased. Os was fully dilated, membranes were still intact, and presentation was normal. Patient got very languid and despondent. Chloroform was administered and delivery completed with forceps at 2 p.m. There was about the ordinary amount of haemorrhage, placentas came away readily. The child was healthy. Patient had been drowsy



drowsy all afternoon and very quiet, sleeping at intervals. About 8 p.m., she thought she saw a light in the room and immediately after was in convulsions. Three fits followed in rapid succession but they were short and not very severe, and patient then developed maniacal symptoms, shouting and struggling to get out of bed and tearing her clothing into shreds. I saw her at 8.30, she was struggling very violently, shouting and throwing her arms about in a wild manner. I immediately injected hypodermically  $\frac{1}{2}$  grain of Morphia. The struggling went on for a few minutes but gradually she quietened and lay down in bed rolling about from side to side and talking incoherently. At 12 midnight she was sick and brought up a quantity of yellowish fluid. She asked for her husband and then remained quiet till 3 a.m., when she got out of bed and passed a large quantity of urine. She was also sick and brought up a small quantity of yellowish fluid. She lay quietly sleeping till 6 p.m., when she again passed urine. On the following day she remembered nothing of what had happened since the child was born. She complained of very severe headache and felt very sore and

and drowsy. Patient looked very pale, face puffy, eyes heavy looking, and there was very marked oedema of feet and legs. She was kept on milk diet and made a slow but good recovery. Temp. 102 first day, normal after the second day. Ammonium Bromide 15 grains was given 4 hourly for headache. Urine was passed freely. It contained a large quantity of albumen. Oedema gradually disappeared and headaches became less severe.

Urine continued to show albumen for 3 months. On enquiring I found that when 15 years of age she had a severe illness and was swollen all over. The Dr said it was "kidney disease." She had been in good health since. The child was fed from bottle. It is now 2 years since the attack. Patient looks quite well. There has been no return of the menstruation.

Two things particularly struck me about the cases I have reported, the unusual frequency--one and two were consecutive cases--and the severity of the type.

The proportion of those attacked with eclampsia vary considerably according to different authors. Herman and Galabin gives 1 in 500, Jelliett from the

the Rotunda reports 1 in 332, while of 227,000 cases collected from continental and British sources there were 635 cases of convulsions giving 1 in 357.

It is not infrequent to meet a doctor who has gone through a large practice for many years without meeting a single case of eclampsia. Occasionally reports are published of a series of eclamptic cases occurring within a very short period for no apparent reason. That of itself suggests the possibility of infection. In my cases the interval between, six weeks and 10 months, and the fact that the patients lived far apart is sufficient to exclude infection. There is one explanation that may be given to account for their frequency, 1 in 20 being the proportion of eclamptic cases in 5 years practice. In our district (South Midlands) a very large proportion of the obstetrical practice among the artisan classes is in the hands of the midwife. The few who seek the services of a medical man, either fear some serious complication of labour, or are patients from the North of England or from Scotland who have been accustomed with a practitioner attending them in confinement. Case I and II were primiparae and the pregnancies illegiti-

illegitimate, both cases had been married less than five months. The relative frequency of primipara and multipara as taken from the numerous reports collected by Herman give an average 79.5% primipara as against 20.5% multipara. It has also been noted that unmarried women suffer more frequently than married. This has been attributed to the shame and mental anxiety associated with their condition. I believe it may also be due to their efforts to hide their condition as long as possible by wearing tight dresses and rarely going out of doors. The severity of type: Case I never regained consciousness after first convulsion. I had no previous knowledge of this patient and with my incomplete notes and the absence of a section I am unable to form an opinion as to the cause. The points for the most part are negative. There had apparently been no warning of any sort. It is not infrequently observed that the prodromal symptoms may be very slight or almost absent. There was no history of "fits" in patient's family and patient had enjoyed good health. Prolonged labour is sometimes given as the exciting cause. The uterine contractions may force a new poison from the



the placenta into the blood of the maternal circulation which is already hypertoxic. In cases I and II there was no sign of any oedema or ascites. Sir James Y. Simpson pointed out that oedema although frequently present was not a necessary accompaniment. Ingersley in a note of 71 cases found oedema absent in 15.

Cases I and II occurring during the puerperium belong to a type which is less frequent than that which occurs during labour. Of about 500 cases collected from different authors, about 250 occurred during labour the other 250 divided about equally between the later months of pregnancy and the puerperium. Bailly<sup>(23)</sup> holds that the order of frequency is pregnancy, labour, and puerperium.

Case II. There were evidences of a toxic condition of blood and renal insufficiency,--despondent state of patient and puffiness under the eyes,--for at least a week and under ordinary circumstances advice would have been sought but the girl was the subject of cruel treatment. There was no previous history of any renal mischief. In both I and II the arterial tension was very high.

Case III. I was in a more favourable position to

to undertake the treatment of this case as I knew the patient and had attended her during a previous illness. Symptoms of toxæmia had been present at least during the later months of pregnancy, but she thought that it was the usual result of her pregnant condition and did not seek advice. This case occurred in Nov., the same time of the year as Case I. There was nothing particular about the weather. Early writers have laid considerable stress on the atmospheric conditions as influencing the onset of the convulsions. They found convulsions more frequent in hot weather than in cold and more particularly when there was "thunder in the air." The season of the year has little effect as statistics show about the same number of cases for each month.

The method of treatment followed in Cases I and II was similar, to stimulate excretory function of skin and empty bowel of fecal matter and allay the irritability of nerve centres by Bromide and Chloral. In Case I, I really saw very little benefit from chloral, chloroform or bleeding. It certainly did not seem to be a case suitable for morphia but might have benefited by the saline

saline injection or by the administration of oxygen.

In Case II the enema had good effect and produced a slight stimulation of the renal functions. There seems considerable diversity of opinion as to whether labour should be induced at once, but after reading recent notes of successful cases treated by rapid dilatation and delivery with forceps, I consider that this would have been possibly the best treatment to adopt in Case II. Delivery is not always followed by a cessation of the fits. Schauta reports that only in 33.5 per cent of the cases do the fits cease after delivery. Case III, the only treatment I adopted was the hypodermic injection of morphine, although morphine is usually contraindicated in renal disease. Some obstetricians go so far as to say they had never used morphia and had no intentions of using it as in albuminuria it is practically fatal. This seemed to me a very suitable case for its use. The mania was of a violent type and there was absence of the extreme condition of coma as seen in the other two cases. Certainly the result was eminently satisfactory. As the prognosis seemed grave I remained in attendance all night and was surprised and pleased to see

see the large amount of urine passed. In practice I have occasionally had my attention drawn by patients to the diuretic effect that administration of morphia had on them, so that it seems unwise to lay down any dogmatic rule for or against the use of morphia. Each case ought to be carefully considered on its own merits.

Stroganoff reports 58 cases of eclampsia without a death. This number he has increased to 113 with 6 deaths. He advocates the use of morphine and chloral and discards chloroform, hot pack, and venesection. He also uses Oxygen. The prognosis of cases of eclampsia associated with mania occurring so soon after delivery is very good, and possibly the chloral treatment might have been attended with equal success. From the one case it can safely be inferred that the administration of morphia did no harm and it was attended with the recovery of the patient.



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